Aetiological influences on continuity and co-occurrence of eating disorders symptoms across adolescence and emerging adulthood

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Abstract

Objective: The role of common and symptom-specific genetic and environmental influences in maintaining eating disorder symptoms across development remains unclear. This study investigates the continuity and change of aetiological influences on drive for thinness, bulimia, and body dissatisfaction symptoms, and their co-occurrence, across adolescence and emerging adulthood.

Method: 2,629 adolescent twins (mean age=15.20, SD=1.95) reported eating disorders symptoms across three waves of data collection. Biometric common pathways model was fitted to estimate genetic and environmental contributions to the continuity of each symptom over time, as well as time- and symptom-specific influences.

Results: Drive for thinness and body dissatisfaction symptoms showed a pattern of high continuity across development, and high correlations with each other, while bulimia symptoms were moderately stable and less associated with the other two symptoms. Latent factors reflecting continuity of each symptom were largely under genetic influence (A_l=.60-.82). New genetic influences contributing to change in the developmental course of symptoms were observed in emerging adulthood. Genetic influences correlated considerably between the three symptoms. Non-shared environmental influences were largely time-and symptom-specific, but some contributed moderately to the continuity across development (E_l=.18-.40). The aetiological overlap was larger between drive for thinness and body dissatisfaction symptoms than with bulimia symptoms.

Discussion: The results provide preliminary evidence that stable as well as newly emerging genetic influences contribute to the co-occurrence of drive for thinness, bulimia, and body dissatisfaction symptoms across adolescence and emerging adulthood. Conversely, environmental influences were less stable and contributed to change in symptoms over time.

Key words: Adolescence; body dissatisfaction; bulimia; development; eating disorders; emerging adulthood; genetics; twins.

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Eating disorders, including anorexia nervosa and bulimia nervosa, increase markedly in adolescence (Hudson, Hiripi, Pope, & Kessler, 2007; Nagl et al., 2016; Patton, Selzer, Coffey, Carlin, & Wolfe, 1999), carrying burden of psychosocial impairment (Kessler et al., 2014; Preti et al., 2009) as well as predicting long-term mental and physical health difficulties and mortality (Crow et al., 2009; Mitchell & Crow, 2006; Zipfel, Giel, Bulik, Hay, & Schmidt, 2015). Importantly, symptoms of eating disorders, such as drive for thinness and body dissatisfaction, also increase in prevalence and are clinically relevant in young people (Herpertz-Dahlmann et al., 2015; Jones, Bennett, Olmsted, Lawson, & Rodin, 2001; Quick & Byrd-Bredbenner, 2013). One of the reasons why eating disorders symptoms are impairing is that they often persist over time (Calzo et al., 2012; Herpertz-Dahlmann et al., 2015; Loth, MacLehose, Bucchianeri, Crow, & Neumark-Sztainer, 2014), although other studies do not find evidence of high continuity of these symptoms in community samples (Glazer et al., 2018; Patton, Coffey, & Sawyer, 2003; Steinhausen, Gavez, & Winkler Metzke, 2005). Therefore, it is crucial to understand the aetiological factors that maintain the eating disorders symptoms across development, in order to inform successful prevention and intervention strategies. As such, the current study investigated the continuity and change of genetic and environmental influences on within- and across-symptom continuity of three eating disorders symptom scales across adolescence and emerging adulthood.

Overlapping aetiology of eating disorders symptoms

Genetic factors play a substantial role in the aetiology of eating disorders across development and in adulthood, including both anorexia nervosa and bulimia nervosa (Bulik, Kleiman, & Yilmaz, 2016; Silberg & Bulik, 2005; Thornton, Mazzeo, & Bulik, 2010), with the remaining variance accounted for by non-shared environmental influences. Shared environmental influences appear to play a role only in

children and younger adolescents (Klump, McGue, & Iacono, 2000). Closely related symptoms, such as restrained eating behaviours and self-induced vomiting, are also heritable (Neale, Mazzeo, & Bulik, 2003; Peterson et al., 2016; Schur, Noonan, Polivy, Goldberg, & Buchwald, 2009). There is a high genetic overlap in the susceptibility to anorexia nervosa and bulimia nervosa (Bulik et al., 2010) and the two disorders, as well as symptoms of these disorders, share many environmental risk factors, including negative life events, weight teasing, and social pressures (Fairweather-Schmidt & Wade, 2015; Haines, Neumark-Sztainer, Eisenberg, & Hannan, 2006; Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004; McKnight Investigators, 2003).

Similarly to other eating disorders symptoms, body dissatisfaction, and related concerns about body weight and shape, showed moderate to high heritability estimates in adolescence (Klump et al., 2010; Klump et al., 2000; Slof-Op't Landt et al., 2008; Suisman et al., 2012; Wilksch & Wade, 2009) and in adulthood (Keski-Rahkonen et al., 2005; Klump et al., 2010; Spanos, Burt, & Klump, 2010; Wade & Bulik, 2007; Wade, Martin, & Tiggemann, 1998), with the remaining variance explained by non-shared environmental influences. Just like in eating disorders, shared environmental influences on weight and shape concerns play a role only in children and younger adolescents (Klump et al., 2010). Twin studies also suggest that a common latent factor is responsible for the genetic influences on body dissatisfaction and disordered eating symptoms (Baker et al., 2009; O'Connor et al., 2017). Taken together, two decades of genetically informative research suggest that core symptoms of eating pathology are underpinned by a comparable degree of genetic vulnerability that may overlap.

Continuity and change of aetiological influences over time

To date, only three longitudinal twin studies have investigated the contribution of genetic and environmental influences to the continuity of eating pathology, all focusing on influences across adolescence. First, Klump, Burt, McGue, and Iacono (2007) investigated genetic and environmental influences on an aggregate score of eating disorder symptoms in 11, 14, and 18 year old twins, and found that stable genetic and shared environmental influences accounted for the symptom continuity, with no

new genetic or shared environmental influences emerging at the latter two ages (genetic and environmental innovation). Second, Fairweather-Schmidt and Wade (2015) investigated eating disorders symptoms at ages 12-15 and 16-19 years, and similarly found stable genetic and shared environmental influences. They also additionally reported significant genetic innovation at the latter time point. Third, Wade et al. (2013) focused on body and shape concerns at ages 12-13, 13-15 and 14-16 years, and observed that both genetic and shared environmental influences contributed to the continuity of this phenotype. They reported genetic and shared environmental innovation only at the second time point, but not at the final one. In all three longitudinal studies, despite contributing to symptom continuity, the overall impact of shared environmental influences became non-significant at later adolescence, while non-shared environmental influences were largely time-specific and contributed to a smaller proportion of continuity over time. Overall, emerging evidence suggests that genetic and shared environmental influences in early adolescence largely underpin the continuity of eating disorder symptoms, with new genetic influences coming online in later stages of adolescence, while the contribution of shared environmental influences declines with age.

Nonetheless, several important developmental questions remain unexplored. First, despite remarkable heterogeneity of eating disorder symptoms, to our knowledge no study has looked at longitudinal aetiological influences on individual symptoms, such as bulimia symptoms or body dissatisfaction. Furthermore, no longitudinal studies to date expanded beyond adolescence, to study the continuity and change in aetiological influences during the transition to emerging adulthood. Emerging adulthood is an important developmental period with regards to new onsets of psychopathology (Arnett, 2014), including eating disorders (Favaro, Caregaro, Tenconi, Bosello, & Santonastaso, 2009; Hudson et al., 2007; Nagl et al., 2016; Udo & Grilo, 2018). Third, no study to date examined how dynamic changes in etiological influences contribute to the co-occurrence of different eating disorder symptoms over time.

Aims

The current study presents the first investigation of genetic and environmental influences on the continuity and co-occurrence of symptoms of drive for thinness, bulimia, and body dissatisfaction, from adolescence into emerging adulthood. In line with the previous findings, we hypothesised that genetic influences will largely underpin continuity of all three symptoms. We also expected to observe moderate genetic innovation, as well as large non-shared environmental innovation, at each time point, including emerging adulthood. Finally, given substantial genetic overlap and moderate non-shared environmental correlations between different eating pathology symptoms, we tentatively hypothesised that both genetic and environmental influences would contribute to the longitudinal co-occurrence of drive for thinness, bulimia, and body dissatisfaction symptom scales.

Methods

Sample

All twin pairs born in Norway between 1988 and 1994 were invited to participate in a prospective, ongoing population-based twin study. The present analyses used information collected from the first three waves of data collection, conducted in 2006, 2008 and 2010 (hereon referred to as times 1-3). At time 1, participants were between 12 and 18 years of age and 57% female. Twin status and addresses were provided by the Norwegian Medical Birth Registry. Upon familial consent, self-report questionnaires were mailed to the twins. The participating sample comprised 1,345 families at time 1, in which at least one family member returned completed survey forms by mail. At time 2, 1,012 families participated, and at time 3, 849 families participated. A total of 1,484 families participated in at least one time point of data collection. The follow-up family assessments participation rates were 68.3% from time 1 to time 2, 68.9% from time 1 to time 3 and 69.4% from time 2 to time 3. Attrition was not associated with baseline eating disorder symptoms. The mean age at Time 1 is 15.20 years old (SD=1.95), 16.90 (SD=1.99) at Time 2, and 19.60 (SD=1.96) at Time 3. The study sample was previously described in detail (Waaktaar & Torgersen, 2012), also see Table 1, Table S1, and Figure S1 for the participant flow chart. The present study focuses on data collected through twin self-reports only.

Measures

Twin zygosity was determined by discriminant analysis based on data from 12 items on twin physical similarity. DNA was obtained from cheek swabs in 15% of the sample. Comparisons of DNA and questionnaire data indicated the misclassification of MZ or DZ based on questionnaire data to be less than 2% (Waaktaar & Torgersen, 2012). The distribution of zygosity groups is presented in Table 1.

Eating disorders symptoms were measured at each time using eleven items from the Eating Disorder Inventory-Revised (EDI-R) (Garner & Olmstedt, 1984). Items were selected through a 2 phase pilot testing on two independent school-based adolescent samples, whereby the items demonstrating the best psychometric properties, i.e. the highest item-to-scale correlations, were chosen. Participants indicated on a 0-5 scale (0-never to 5-always) how often in the preceding 12 months they endorsed three drive for thinness subscale symptoms (e.g. "*I am terrified of gaining weight*"), four bulimia subscale symptoms *(e.g. "I have gone on eating binges where I have felt that I could not stop"*), and four body dissatisfaction subscale symptoms (e.g. *"I think that my stomach is too big"*). The items were summed to create three eating disorders symptom subscales. The EDI-R is commonly used in adolescent and young adult samples (Klemchuk, Hutchinson, & Frank, 1990; Shore & Porter, 1990), is translated and validated in Norwegian (Rosenvinge, Sundgot Borgen, & Börresen, 1999), and demonstrated sound internal consistencies in the current sample (α=.74-.90, Table 1). The three factor structure was further corroborated using confirmatory factor analyses at each time point, and showed adequate model fit (CFI=.938-.949; TLI=.916-.932; RMSEA=.093-.103; SRMR=.055-.066, see Table S2). Finally, each subscale demonstrated significant time invariance (Table S3).

Analyses

Descriptive statistics and other phenotypic analyses were calculated using Stata (StataCorp., 2007). A repeated measures analysis of variance (ANOVA), including one factor (time), was conducted on mean

eating disorders symptoms to establish whether they significantly increased across time. The analyses controlled for non-independence of the data by randomly selecting only one member of the twin pair.

Twin studies assess the similarity between monozygotic (MZ, sharing 100% of their genes) and dizygotic (DZ, sharing on average 50% of their segregating genes) twins within a pair. Differences in correlations within MZ and DZ twin pairs allow estimations of the influences due to: additive genetics (A, the heritability of a trait), dominant genetics (D, interactions between alleles, which are not transmitted from parents to offspring), shared environment (C, non-genetic factors that contribute to similarity between twins) and non-shared environment (E, non-genetic factors that contribute to differences between twins, and this parameter also includes measurement error). The estimated, latent influences tell us how much of the construct's variance at the population level is due to genetics (additive, dominant) and environment (shared, non-shared), however they cannot provide information about which specific genes or experienced play a role in the etiology. For more details on twin modelling methods see (Plomin, DeFries, Knopik, & Neiderhiser, 2013; Rijsdijk & Sham, 2002).

Models were fitted using a structural equation modeling package for genetically informative data, OpenMx (Boker et al., 2011) within R (www.R-project.org (TeamRDC, 2010)). Models were fitted using full information maximum likelihood. To assess how well models fit the data, the main fit statistic was minus twice the log likelihood (-2LL) of the observations, a relative measure of fit, with differences in -2LL between models distributed as χ^2. We also used Akaike's Information Criterion, with lower values suggesting a better fit. If the difference between the AIC of two models was less than 10, the more parsimonious model was selected (Wagenmakers & Farrell, 2004). To examine the overall fit of the genetic model we compared its -2LL and AIC to that of a saturated model (which fully describes data using the maximum number of free parameters, estimating variances, covariances and means for the raw data to get a baseline index of fit). Likewise, we used the χ^2 difference tests and the AIC to test the fit of nested submodels. Finally, we also estimated likelihood-based confidence intervals.

Univariate genetic analyses were conducted to estimate proportion of genetic and environmental influences on all individual variables, at each time point. For multivariate analyses, a common pathway model was chosen a priori and only A and E influences were fitted. In this model, a common phenotypic latent factor influencing observed variables is derived, and genetic and environmental influences on this latent phenotypic factor are estimated. For current analyses, we modelled three phenotypic latent factors, each one loading on the same symptom assessed at three time points, e.g. the latent drive for thinness factor loaded on observed drive for thinness at times 1, 2 and 3. Thus, each latent factor captured the within-symptom continuity of the given symptom across all three time points. Genetic (A_l) and environmental (E_l) influences on each latent factor were derived to assess the contribution of these factors to the within-symptom continuity of each symptom. Of note, E_l is free from time-specific measurement error but not from shared measurement error. Since we had three latent phenotypic factors, we were able to correlate genetic and environmental influences on each factor (r_{Al} and r_{El}). These correlations represent genetic and environmental overlap in the co-occurrence of these symptoms over time (across-symptom continuity). The genetic overlap can be due to allelic pleiotropy (same genes affect several traits) as well as mediated pleiotropy (allele influences several traits, but its effects on some traits are secondary to more direct effects on the other traits). The environmental overlap can be in part due to shared measurement error. We also calculated the proportion of the phenotypic correlations between the latent factors that is due to genetic and environmental influences.

Any remaining variance in observed variables that was not explained by the latent phenotypic factor was then calculated as variable-specific genetic and environmental influences (A_s and E_s). These residual influences capture factors that do not operate across 3 time points. These variable-specific factors include genetic and environmental influences that emerge at later time points (genetic innovation at times 2 and 3). These influences are allowed to correlate with influences on all other variables at the same time point (r_{As} and r_{Es}), capturing concurrent associations between them. For more details on this particular model, see Waszczuk, Zavos, Gregory, and Eley (2016).

Results

Phenotypic results

Mean scores on each eating disorders symptom at each time point are presented in Table 1. There was a moderate main effect of time on drive for thinness symptoms scores, F=(1.87, 1041.69, Huynh-Feldt correction)= 50.84, p<.001, η_p^2=.08. Body dissatisfaction also increased significantly across development, F=(1.84, 1029.19, Huynh-Feldt correction)= 10.33, p<.001, η_p^2=.02, which indicates a small effect size. Post-hoc analyses revealed that only time 1 body dissatisfaction score was significantly different from the two subsequent scores. Bulimia symptoms did not increase significantly over time.

The concurrent and longitudinal correlations between the variables across the three time points are presented in Table 2. There was a larger concurrent association between drive for thinness and body dissatisfaction symptoms (r=.83-.84), than between bulimia and the other two eating disorders symptoms (r=.46-.59). Drive for thinness and body dissatisfaction symptoms showed high continuity across the three time points (r=.55-.75). In contrast, bulimia symptoms showed only moderate continuity (r=.34-50). The longitudinal across-symptoms correlations between the three eating disorders symptoms were generally moderate to high (r=.22-.65), with somewhat larger across-symptoms continuity between drive for thinness symptoms and body dissatisfaction, than with bulimia. Longitudinal within-symptom correlations were generally comparable to across-symptoms correlations, and tended not to decrease markedly at longer time intervals (time 1 to time 3). See Table S4 for phenotypic correlations by zygosity.

Twin modelling results

In univariate analyses, the ADE models fitted the data best (Table S5). However, large samples are needed to reliably distinguish between A and D effects (Rietveld, Posthuma, Dolan, & Boomsma, 2003). As such, the AE models were fitted, where A should be interpreted as broad sense heritability comprising

of both additive and dominant genetic influences in all subsequent analyses (see Table S6 for model fit statistics and submodel comparisons). Both drive for thinness and body dissatisfaction were characterized by large genetic influences (A=.56-61 and .65-.58, respectively), with remaining variance explained by non-shared environmental effects (Table 3). Bulimia symptoms were under significantly lower, moderate genetic influences (A=.31-.49).

In the common pathway model, the phenotypic latent factor, and the variable-specific influences, jointly explain the total variance in each variable. Latent phenotypic factors, which capture within-symptom continuity, accounted for 24-86% of the variance in each variable (L^2, see Figure 1 and Table S7 footnote). The latent factors were generally highly correlated (r_{phl}=.63-.91, Table 4a). Latent phenotypic factors were largely influenced by genes (A_l=.60-.82), and by modest to moderate non-shared environmental influences (E_l=.18-.40). Genetic influences on latent factors correlated considerably across the three factors (r_{Al}=.72-.92), and the non-shared environmental overlap between them were also high (r_{El}=.47-.88). Phenotypic correlations between the three stable factors were largely due to genetic influences, and to lesser extent due to non-shared environmental influences, see Figure 2. This indicates common genetic and environmental contributions to the co-occurrence of different eating disorders symptoms across development (across-symptoms continuity).

Since most of the genetic influences acted via the latent factors, the residual genetic influences were small (A_s=.00-.16, see Figure 1 and Table S7). The variable-specific genetic influences that capture genetic innovation were only significant at time 3 ($A_{s,\ time\ 3}$=.11-.13). Conversely, residual non-shared environmental influences were generally moderate (E_s=.13-.61) and there was significant non-shared environmental innovation at both times 2 and 3. The concurrent phenotypic, genetic, and environmental correlations between the variable-specific, residual influences varied widely (Table 4b). Both latent and time-specific genetic and environmental influences tended to correlate significantly more between drive for thinness and body dissatisfaction, than with bulimia symptoms.

Finally, sensitivity analyses were conducted to test whether the results replicate in a subgroup of participants who fell into non-overlapping age groups across the three time points (time 1=14-15 years, time 2=16-17 years, time 3=18-19 years). The broad sense heritability and environmental influences were not significantly different from those obtained in the whole cohort (Table S8).

Discussion

To our knowledge, the current study is the first to investigate how genetic and environmental influences contribute to the development of three co-occurring eating disorders symptoms: drive for thinness, bulimia, and body dissatisfaction. The results indicate similar continuity of each of these symptoms across adolescence and emerging adulthood, which was underpinned by largely non-specific and stable genetic influences. Genetic innovation was observed only when participants reached emerging adulthood. Most environmental influences were time and symptom-specific, and generally contributed to change in symptoms over time. Nonetheless, a small proportion of stable non-shared environmental influences played a role in the symptom continuity and co-occurrence across development.

Within-symptom continuity

All symptoms were characterised by considerable within-symptom continuity, with drive for thinness and body dissatisfaction symptoms showing somewhat higher continuity than that of bulimia symptoms. The persistence of eating disorders symptoms across adolescence and emerging adulthood is in line with previous findings (Calzo et al., 2012; Herpertz-Dahlmann et al., 2015; Loth et al., 2014), although not all previous studies reported high symptom continuity (Glazer et al., 2018; Patton et al., 2003; Steinhausen et al., 2005). The severity of both drive for thinness and body dissatisfaction symptoms increased significantly across development, while bulimia symptoms remained at the same level.

The within-symptom continuity of each symptom was mostly due to stable genetic influences, as expected based on previous studies (Fairweather-Schmidt & Wade, 2015; Klump et al., 2007; Wade et al., 2013). The current study was underpowered to distinguish between additive and dominant genetic

influences and as such stable genetic effects reported should be interpreted as a broad-sense heritability. Non-shared environmental influences were generally time-specific and there was significant innovation at each time point, which means that environmental contribution was towards the change in symptoms over time, which is also in line with previous studies. Furthermore, we found that there were new genetic influences in emerging adulthood. The current study is the first to investigate the genetic continuity in eating disorders symptoms beyond adolescence and found that new genetic influences emerge at this important developmental stage, which might reflect the continued onsets of eating disorders in early 20s (Favaro et al., 2009; Hudson et al., 2007; Nagl et al., 2016; Udo & Grilo, 2018). Taken together, these developmentally dynamic etiological effects can contribute to change in the course of eating disorders symptoms form adolescence to emerging adulthood. Notably, chronic bulimia symptoms were significantly less influenced by genetic factors than either chronic drive for thinness or body dissatisfaction symptoms, with non-shared environmental influences playing a greater role in maintaining bulimia symptoms.

Across-symptoms continuity

Continuity across the symptom scales was significant, which was largely explained by genetic correlations, both between stable genetic influences that contribute to the continuity of each disorder symptom, and between the time-specific genetic influences that capture genetic innovation. This suggests that genetic influences on both stable and time-specific symptoms have broad effects, i.e. contributing to all three types of eating disorders symptoms. Notably, both the longitudinal and time-specific genetic correlation between drive for thinness and body dissatisfaction symptoms was significantly higher than with bulimia symptoms. This suggests that drive for thinness and body dissatisfaction symptoms may be underpinned by more common genetic vulnerability than bulimia symptoms in young people.

These results of genetic continuity carry implications for molecular genetic studies of eating disorders (Boraska et al., 2014; Duncan et al., 2017; Liu, Study, Kelsoe, & Greenwood, 2016) They provide

preliminary support for broadening phenotypes included in molecular genetic studies to encompass a wide range of eating pathology phenotypes, to increase power to detect shared susceptibility loci (Smoller et al., 2018) Furthermore, given substantial stable genetic influences across adolescence and young adulthood, molecular genetic studies might not benefit from stratifying developmental samples by age. Identifying specific genes or polygenic risk scores may in turn inform precision medicine, for example by using genetic markers to predict disease risk or treatment response.

Alongside genetic influences, a small proportion of environmental influences was found to play a significant role in the maintenance of eating disorders symptoms. These stable environmental influences contributed significantly to the longitudinal co-occurrence of the three symptom scales. While twin studies cannot provide information about which environmental influences contributed the symptoms continuity without directly measuring exposures and experiences, these may include transdiagnostic risk factors identified in previous research, such as the sociocultural effects, e.g. the impact of the media and the peers, as well as sexual abuse and other adverse experiences (Fairweather-Schmidt & Wade, 2015; Haines et al., 2006; Jacobi et al., 2004; McKnight Investigators, 2003; Wade, Gillespie, & Martin, 2007). Future twin studies should include measures of environmental risk factors, and identify which of them operate in the stable and broad manner to inform transdiagnostic interventions and prevention strategies in eating disorders (Fairburn et al., 2009; Fairburn, Cooper, & Shafran, 2003). Finally, as with longitudinal genetic effects, stable non-shared environmental influences overlapped more highly between drive for thinness and body dissatisfaction symptoms than with bulimia symptoms.

The reasons for the overall pattern of drive for thinness and body dissatisfaction showing closer etiological links than bulimia symptoms remain to be investigated. One potential explanation might be different personality traits characterizing these symptoms. Specifically, the former two symptoms share traits such as perfectionism, achievement striving, and rigidity (Chang, Ivezaj, Downey, Kashima, & Morady, 2008; Hewitt, Flett, & Ediger, 1995; Luo, Forbush, Williamson, Markon, & Pollack, 2013), in line with personality findings for anorexia nervosa (Bardone-Cone et al., 2007; Wonderlich, Lilenfeld,

Riso, Engel, & Mitchell, 2005). In contrast, bulimia symptoms are associated with personality traits related to disinhibition and impulsiveness, especially in response to negative affect (Fischer, Smith, & Cyders, 2008), which is again in line with personality findings for bulimia nervosa (Danner, Sternheim, & Evers, 2014; Dawe & Loxton, 2004; Vitousek & Manke, 1994).

Limitations

The genetically-informative, representative sample and multiple time points are strengths of the current study. However, a number of limitations are noteworthy. First, our analyses used only self-report symptom scales and the results should be replicated in clinical samples and using lifetime diagnostic interviews. This approach was taken because clinical levels of eating disorders are very rare in general adolescent population and questionnaires capture subthreshold symptoms of these disorders, which also constitute important markers of psychopathology (Jones et al., 2001; Le Grange et al., 2006; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011; Tozzi et al., 2005). Common mental disorders are now considered to be the extremes of quantitative traits (Insel et al., 2010; Kotov et al., 2017; Plomin, Haworth, & Davis, 2009) and there is evidence that differently defined eating problems have the same aetiology (Bulik et al., 2016; Thornton et al., 2010). Second, although univariate results suggested that a significant proportion of genetic influences consists of dominant genetic effects, we were underpowered to distinguish them from the additive genetic effects, and as such modelled broad genetic effects. Furthermore, by selecting the best fitting ADE models, the study was unable to assess the role of shared environmental effects. However, the pattern of MZ and DZ correlations suggests that shared environment was unlikely to make important contributions to the current phenotypes. Third, there was attrition in the sample, which was not associated with baseline eating disorders symptoms, but may be systematically related to other characteristics of the participants who self-select to remain in the study and as such could bias the results. Although attrition bias might complicate estimation of trait prevalence, it is unlikely to affect the estimation of between trait associations (Wolke et al., 2009). Fourth, overlapping age ranges limit our ability to draw conclusions about the correspondence of time-specific influences to particular

ages. However, the majority of the sample fell within non-overlapping age ranges at each time point, and the findings were very similar when analyses were restricted to non-overlapping age groups. A replication in independent samples is necessary to confirm the findings of developmental changes in the etiology of eating disorder symptoms, in particular the genetic innovation in emerging adulthood.

Fifth, we did not measure other eating disorders symptoms such as purging, and future research should extend our findings to a wider range of eating disorders symptoms. Sixth, we also did not measure specific environmental exposures, therefore cannot elaborate on which experiences contributed to the continuity vs change of symptoms in the current study. Future twin studies of eating pathology should include a wider range of environmental measures, such as stressful life events interviews, to better address this issue. Seventh, lower reliability and higher non-shared environmental influences, thus potentially higher measurement error, on bulimia symptoms could in part account for its lower continuity, and lower associations with other eating disorders symptoms. Last, there are limitations inherent to the twin design and statistical modelling of twin data, discussed comprehensively elsewhere (Plomin et al., 2013). These have minimal and contrasting effects on parameter estimates which should be taken as indicative rather than absolute, and all structural equation models are approximate and for this reason should be interpreted with caution (Tomarken & Waller, 2005). It is also worth noting that the model selection was tailored to the current research questions, but fitting alternative models could lead to different conclusions.

Conclusions

Our results suggest that continuity and developmental co-occurrence of drive for thinness, bulimia, and body dissatisfaction symptoms across adolescence and emerging adulthood is underpinned largely by stable genetic influences, while non-shared environmental effects tend to be more transient and contribute to change in symptoms over time. The results inform the current understanding of eating disorders symptoms' etiology and have implications for future molecular genetics research. Future studies need to

continue examining how the risk and maintenance factors for eating disorders operate across development to inform the translation of the etiological findings to clinical practice.

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Tables

Table 1 – Descriptive statistics

Time	Mean age (SD)	Age range	N	MZ/DZ	Symptom	Alpha	Mean (SD)-total sample	Mean (SD)-MZ twins	Mean (SD DZ twins
1	15.20 (1.95)	12-18 years	2,629	999/1630	Drive for thinness	.90	2.75 (3.86)	2.68 (3.81)	2.80 (3.90
					Bulimia	.79	1.05 (2.34)	1.02 (2.45)	1.07 (2.27
					Body dissatisfaction	.88	4.96 (4.81)	4.82 (4.47)	5.06 (4.86
2	16.90 (1.99)	14-20 years	1,891	711/1180	Drive for thinness	.90	3.40 (4.06)	3.53 (4.08)	3.32 (4.06
					Bulimia	.74	1.24 (2.43)	1.32 (2.57)	1.20 (2.35
					Body dissatisfaction	.88	5.62 (4.96)	5.81 (5.07)	5.51 (4.89
3	19.60 (1.96)	16-22 years	1,453	557/896	Drive for thinness	.89	4.20 (4.22)	4.22 (4.17)	4.21 (4.27
					Bulimia	.74	1.34 (2.48)	1.24 (2.48)	1.41 (2.49
					Body dissatisfaction	.87	5.64 (5.07)	6.82 (5.13)	6.84 (5.21

Notes:

MZ – monozygotic, DZ – dizygotic. N is in individuals. Drive for thinness symptoms score range is 0-15, Bulimia and Body dissatisfaction score range is 0-20.

Means could be equated across zygosity, except for body dissatisfaction at time 1. Variances could be equated across zygosity, except for bulimia at times 1 and 2. See Table S6 for constrained models fit statistics.

Attrition was not associated with baseline eating disorder symptoms.

Table 2 – Phenotypic Pearson's correlations

		Time 1			Time 2			Time 3	
		Drive for thinness	Bulimia	Body dissatisfaction	Drive for thinness	Bulimia	Body dissatisfaction	Drive for thinness	Bulimia
Time 1	Bulimia	.58 (.54-.61)							
	Body dissatisfaction	.84 (.82-.86)	.52 (.48-.56)						
Time 2	Drive for thinness	.68 (.64-.71)	.35 (.29-.41)	.65 (.61-.69)					
	Bulimia	.40 (.34-.45)	.39 (.33-.45)	.36 (.30-.42)	.54 (.49-.58)				
	Body dissatisfaction	.62 (.58-.66)	.33 (.27-.39)	.71 (.68-.74)	.83 (.81-.85)	.46 (.41-.51)			
Time 3	Drive for thinness	.55 (.49-.60)	.25 (.18-.32)	.55 (.49-.60)	.70 (.66-.74)	.41 (.34-.47)	.65 (.60-.69)		
	Bulimia	.43 (.37-.49)	.34 (.27-.41)	.41 (.34-.47)	.41 (.34-.47)	.50 (.44-.56)	.42 (.35-.48)	.59 (.54-.64)	
	Body dissatisfaction	.51 (.45-.57)	.22 (.15-.29)	.61 (.56-.66)	.65 (.60-.69)	.35 (.28-.42)	.75 (.71-.78)	.83 (.81-.85)	.53 (.48-.58)

Notes:

The correlations control for the non-independence of data.

Table 3 – Univariate results

		A	E
Time 1	Drive for thinness	.61 (.56-.66)	.39 (.34-.44)
	Bulimia	.31 (.24-.37)	.69 (.63-.76)
	Body dissatisfaction	.68 (.63-.72)	.32 (.28-.37)
Time 2	Drive for thinness	.58 (.51-.64)	.42 (.36-.49)
	Bulimia	.32 (.24-.40)	.68 (.60-.76)
	Body dissatisfaction	.65 (.59-.70)	.35 (.30-.41)
Time 3	Drive for thinness	.56 (.48-.64)	.44 (.36-.52)
	Bulimia	.39 (.29-.49)	.61 (.51-.71)
	Body dissatisfaction	.68 (.61-.74)	.32 (.26-.39)

Notes:

Heritability (A) does not distinguish between additive and dominant genetic effects, and as such should be interpreted as broad-sense heritability. For full univariate ADE results, see Table S5 in the Appendix.

Table 4 – Common pathway model results: phenotypic, genetic and non-shared environmental correlations between (a) latent factors and (b) time-specific influences

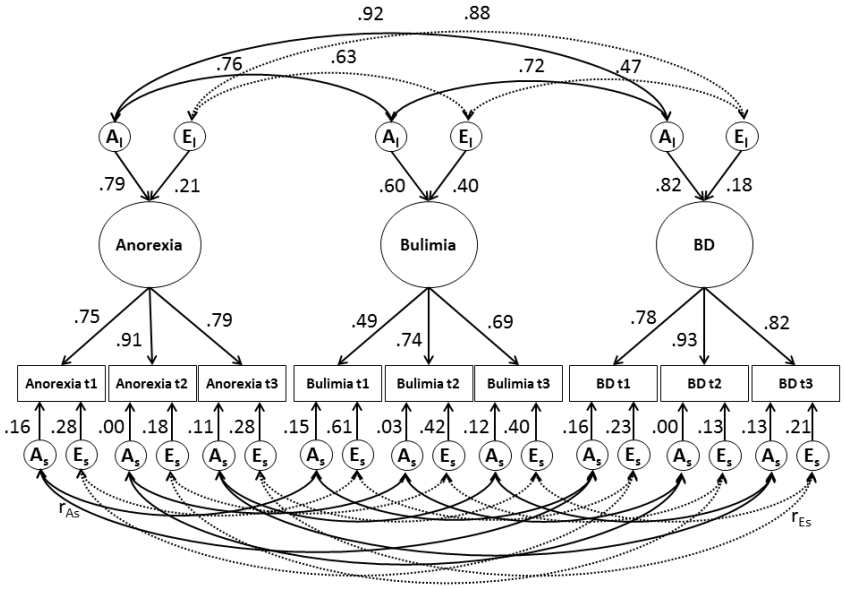
	Drive for thinness	Bulimia	Drive for thinness	Bulimia	Drive for thinness	Bulimia
(a) Correlations between latent factors						
	Phenotypic correlations (r_{phl})		Genetic correlations (r_{Al})		Environmental correlations (r_{El})	
Bulimia	.71 (.67-.74)		.77 (.70-.83)		.63 (.49-.74)	
Body dissatisfaction	.91 (.90-.92)	.63 (.59-.67)	.92 (.90-.94)	.72 (.65-.80)	.88 (.82-.92)	.47 (.32-.61)
(b) Within-time correlations between time-specific influences						
	Phenotypic correlations (r_{phs})		Genetic correlations (r_{As})		Environmental correlations (r_{Es})	
Time 1						
Bulimia	.50 (.47-.54)		.68 (.51-.85)		.45 (.38-.51)	
Body dissatisfaction	.70 (.67-.73)	.45 (.41-.49)	.83 (.74-.90)	.67 (.49-.85)	.62 (.56-.58)	.37 (.30-.45)
Time 2						
Bulimia	.29 (.20-.37)		N/A[a]		.29 (.19-.38)	
Body dissatisfaction	.46 (.36-.53)	.10 (.00-.20)	N/A[a]	N/A[a]	.46 (.36-.54)	.11 (.00-.22)
Time 3						
Bulimia	.46 (.41-.52)		.52 (.22-.83)		.44 (.33-.55)	
Body dissatisfaction	.70 (.66-.73)	.33 (.27-.40)	.80 (.68-.92)	.31 (.00-.61)	.66 (.58-.72)	.35 (.22-.47)

Notes: Heritability (A) does not distinguish between additive and dominant genetic effects, and as such should be interpreted as broad-sense heritability.

[a] Genetic influences specific to time 2 were non-significant and very small in magnitude. For this reason genetic correlations between time 2 influences are non-significant and cannot be reliably estimated.

Figure Captions

Figure 1 - Common pathway model

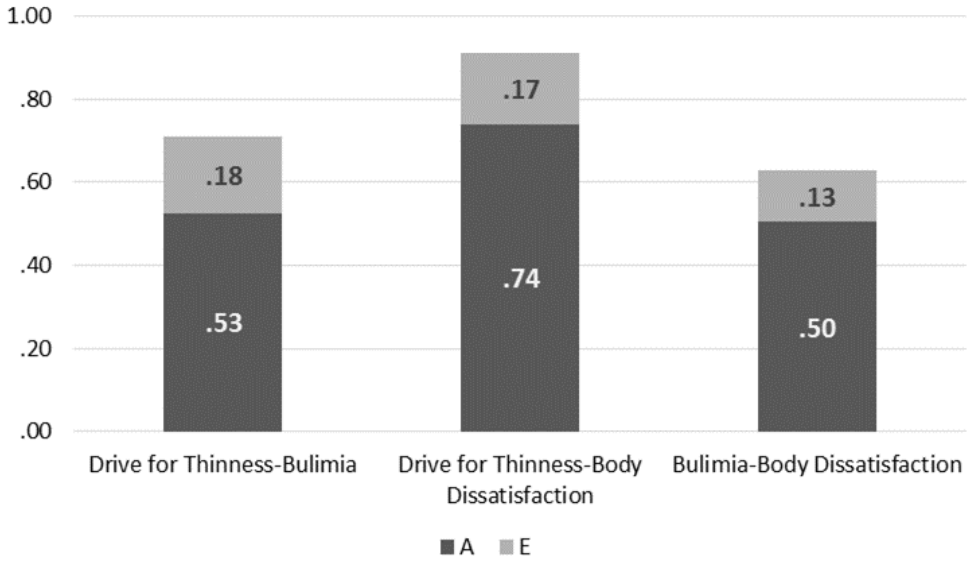


Notes: DfT – Drive for thinness; BD – Body dissatisfaction;

Within-time correlations between time-specific influences are presented in Table 4. For 95% CIs, see Table 4 and Table S7.

Heritability (A) does not distinguish between additive and dominant genetic effects, and as such should be interpreted as broad-sense heritability.

Figure 2 - Proportion of phenotypic correlation between stable factors due to genetic (A) and environmental (E) influences



SUPPLEMENTARY MATERIALS

Table S1 –(a) Combination of time point participation, and (b) total number of pairs at each time point

Combination of time point participation	
Time points	N Pairs
1, 2, 3	654
1, 2	264
1, 3	103
2, 3	47
1	324
2	47
3	45
Together	1484
Total number of pairs at each time point	
Time point	N Pairs
1	1345
2	1012
3	849

Table S2 – Confirmatory Factor Analyses results

	Factor loadings		
	Time 1	Time 2	Time 3
Factor 1 - Drive for thinness			
Think about dieting	.93	.93	.94
Preoccupied with the desire to be thinner	.91	.91	.91
Terrified of gaining weight	.76	.74	.73
Factor 2- Bulimia			
Gone on eating binges, felt that could not stop	.69	.70	.69
Eat and drink in secrecy	.66	.70	.70
Eat moderately in front of others, stuff myself when they're gone	.65	.63	.71
Thought of trying to vomit in order to lose weight	.75	.67	.59
Factor 3 – Body dissatisfaction			
Think that thighs are too large	.87	.87	.86
Think that stomach is too big	.89	.90	.90
Feel satisfied with body shape (R)	.50	.55	.59
Think that buttocks are too large	.74	.72	.71
	Model fit statistics		
CFI	.949	.943	.938
TLI	.932	.923	.916
RMSEA (CIs)	.093 (.088-.098)	.097 (.091-.103)	.103 (.096-.11)
SRMR	.055	.066	.066

Note - Conventional rule-of-thumb guidelines suggest that a fit is acceptable if CFI and TLI are above .90, and RMSEA and SRMR are below .10 (Hu & Bentler, 1998, 1999; Kline, 2011; Marsh, Hau, & Wen,

2004). The fit statistics for the current confirmatory factor analysis jointly demonstrate that the three factor structure fits the data well at each time point, with the exception of RMSEA at times 2 and 3.

Table S3 - Time invariance models fit statistics

Drive for thinness				
	CFI	RMSEA	ΔCFI	ΔRMSEA
Configural model	.986	.069		
Constrained loadings	.986	.065	.001	.004
Constrained intercepts	.982	.068	.004	.003
Constrained means	.959	.099	.023	.031
Bulimia				
	CFI	RMSEA	ΔCFI	ΔRMSEA
Configural model	.930	.075		
Constrained loadings	.918	.076	.012	.001
Constrained intercepts	.915	.073	.003	.003
Constrained means	.913	.073	.002	<.001
Body dissatisfaction				
	CFI	RMSEA	ΔCFI	ΔRMSEA
Configural model	.959	.084		
Constrained loadings	.959	.079	<.001	.005
Constrained intercepts	.947	.086	.012	.006
Constrained means	.936	.093	.011	.007

Notes:

CFI = Comparative Fit Index; RMSEA = root mean square error of approximation

Three types of time invariance were tested. As a base model, configural invariance postulates the same factor structure (number of factors and pattern of loadings) across methods, but does not impose any formal equality constraints. Constrained loadings model fixed factor loadings to be the same at each time point, testing weak invariance. Constrained threshold loadings additionally fixed item intercepts to test strong invariance, while the constrained means model constrained factor means at each time point. ΔCFI > .010 and ΔRMSEA > .015 between the two most proximal models indicate significant deterioration of the fit (Chen, 2007; Cheung & Rensvold, 2002).

Table S4 – MZ and DZ within-twin pair correlations

MZ										
Time		1			2			3		
		Drive for thinness	Bulimia	Body diss.	Drive for thinness	Bulimia	Body diss.	Drive for thinness	Bulimia	Body diss.
1	Drive for thinness	.63 (.58-.68)								
	Bulimia	.33 (.25-.41)	.35 (.27-.42)							
	Body dissatisfaction	.60 (.54-.65)	.32 (.24-.40)	.70 (.65-.74)						
2	Drive for thinness	.53 (.45-.60)	.30 (.20-.40)	.54 (.46-.61)	.61 (.54-67)					
	Bulimia	.28 (.18-.38)	.27 (.17-.38)	.28 (.18-.38)	.33 (.23-.42)	.35 (.26-.43)				
	Body dissatisfaction	.45 (.36-.53)	.26 (.16-.36)	.58 (50-.65)	.57 (.50-.64)	.30 (.20-.39)	.67 (.61-.73)			
3	Drive for thinness	.41 (.30-.51)	.16 (.04-.28)	.42 (.31-.52)	.50 (.40-.59)	.20 (.07-32)	.49 (.38-.58)	.59 (.51-.66)		
	Bulimia	.24 (.12-.35)	.23 (.11-.34)	.23 (.11-.34)	.25 (.12-.37)	.18 (.05-.30)	.28 (.16-.40)	.34 (.23-.44)	.44 (.34-.53)	
	Body dissatisfaction	.40 (.29-.50)	.14 (.02-.26)	.51 (.41-.60)	.48 (.37-.57)	.25 (.12-.37)	.60 (.51-.68)	.55 (.46-.63)	.28 (.16-.38)	.68 (.61-.74)
DZ										
Time		1			2			3		
		Drive for thinness	Bulimia	Body diss.	Drive for thinness	Bulimia	Body diss.	Drive for thinness	Bulimia	Body diss.
1	Drive for thinness	.21 (.15-.27)								
	Bulimia	.12 (.05-.19)	.11 (.04-.18)							
	Body dissatisfaction	.18 (.11-.25)	.11 (.04-.17)	.20 (.13-.27)						
2	Drive for thinness	.16 (.08-.24)	.06 (.00-.14)	.16 (.08-.24)	.17 (.09-.25)					
	Bulimia	.08 (.00-.16)	.07 (.00-.14)	.08 (.00-.16)	.08 (.00-.16)	.10 (.02-.18)				

	Body dissatisfaction	.14 (.05-.22)	.05 (.00-.13)	.17 (.09-.25)	.19 (.11-.27)	.11 (.03-.19)	.22 (.15-.30)			
3	Drive for thinness	.14 (.04-.23)	.02 (.00-.11)	.14 (.04-.23)	.16 (.06-.26)	.07 (.00-.17)	.17 (.07-.27)	.13 (.04-.22)		
	Bulimia	.02 (.00-.12)	.02 (.00-.12)	.08 (.00-.18)	.08 (.00-.18)	.08 (.00-.18)	.11 (.01-.21)	.04 (.00-.13)	.04 (.00-.13)	
	Body dissatisfaction	.18 (.09-.27)	.08 (.00-.18)	.20 (.11-.29)	.17 (.07-.27)	.06 (.00-.15)	.21 (.11-.30)	.14 (.04-.23)	.08 (.00-.17)	.22 (.13-.34)

Table S5 – Univariate results – full ADE models

		A	D	E
Time 1	Drive for thinness	.22 (.00-.48)	.42 (.15-.67)	.36 (.32-.41)
	Bulimia	.17 (.00-.36)	.15 (.00-.38)	.68 (.61-.75)
	Body dissatisfaction	.11 (.00-.37)	.59 (.33-.74)	.29 (.26-.34)
Time 2	Drive for thinness	.07 (.00-.40)	.55 (.21-.67)	.38 (.33-.45)
	Bulimia	.14 (.00-.39)	.20 (.00-.42)	.66 (.58-.75)
	Body dissatisfaction	.24 (.00-56)	.43 (.10-.71)	.33 (.28-.39)
Time 3	Drive for thinness	.00 (.00-.37)	.61 (.52-.67)	.39 (.33-.48)
	Bulimia	.00 (.00-.29)	.44 (.13-.53)	.56 (.47-.66)
	Body dissatisfaction	.24 (.00-.62)	.46 (.07-.74)	.30 (.25-.37)

Table S6 – Univariate model fit statistics

					Fit against saturated model			Fit against ADE model			
Variable	**Time**		**-2LL**	**df**	**chi-sq**	**diff df**	**p-val**	**chi-sq**	**diff df**	**p-val**	**AIC**
Drive for thinness	1	Saturated model	14271.64	2619							9033.64
		Constrained means	14272.21	2621	.57	2	.75				9030.21
		Constrained variance	14274.55	2621	2.91	2	.23				9032.55
		ACE	14285.33	2625	13.69	6	.03				9035.32
		ADE	14276.04	2625	4.40	6	.62				9026.04
		AE	14285.33	2626	13.69	7	.06	9.29	1	>.01	9033.33
		E	14564.31	2627	292.67	8	>.01	288.27	2	>.01	9310.31
	2	Saturated model	11848.91	2619							6610.91
		Constrained means	11849.28	2621	.37	2	.83				6607.28
		Constrained variance	11860.52	2621	11.61	2	>.01				6618.52
		ACE	11864.24	2625	15.33	6	.02				6614.24
		ADE	11863.42	2625	14.51	6	.02				6613.42
		AE	11864.24	2626	15.33	7	.03	.82	1	.37	6612.24
		E	11936.72	2627	87.81	8	>.01	73.30	2	>.01	6682.72
	3	Saturated model	15350.88	2618							10114.88
		Constrained means	15352.41	2620	1.53	2	.47				10112.41
		Constrained variance	15352.66	2620	1.78	2	.41				10112.66
		ACE	15374.99	2624	24.11	6	>.01				10126.99

		ADE	15354.90	2624	4.02	6	.67			10106.90
		AE	15374.99	2625	24.11	7	>.01	20.09	1	>.01	10124.99
		E	15719.46	2626	368.58	8	>.01	364.56	2	>.01	10467.46
Bulimia	1	Saturated model	10488.65	1881							6726.65
		Constrained means	10492.17	1883	3.52	2	.17				6726.17
		Constrained variance	10489.83	1883	1.18	2	.55				6723.83
		ACE	10505.20	1887	16.55	6	.01				6731.21
		ADE	10494.89	1887	6.24	6	.40				6720.89
		AE	10505.20	1888	16.55	7	.02	10.31	1	>.01	6729.21
		E	10670.78	1889	182.13	8	>.01	175.89	2	>.01	6892.78
	2	Saturated model	8659.38	1881							4897.38
		Constrained means	8660.56	1883	1.18	2	.55				4894.56
		Constrained variance	8670.94	1883	11.56	2	>.01				4904.94
		ACE	8675.73	1887	16.35	6	.01				4901.73
		ADE	8674.82	1887	15.44	6	.02				4900.82
		AE	8675.73	1888	16.35	7	.02	.91	1	.34	4899.73
		E	8725.15	1889	65.77	8	>.01	50.33	2	>.01	4947.15
	3	Saturated model	11188.92	1881							7426.92
		Constrained means	11190.86	1883	1.94	2	.38				7424.86
		Constrained variance	11190.89	1883	1.97	2	.37				7424.89
		ACE	11199.95	1887	11.03	6	.09				7425.95

		ADE	11193.20	1887	4.28	6	.64			7419.20
		AE	11199.95	1888	11.03	7	.14	6.75	1	.01	7423.95
		E	11424.65	1889	235.73	8	>.01	231.45	2	>.01	7646.65
Body dissatisfaction	1	Saturated model	8192.35	1443							5306.35
		Constrained means	8201.93	1445	9.58	2	.01				5311.93
		Constrained variance	8194.09	1445	1.74	2	.42				5304.09
		ACE	8213.69	1449	21.34	6	>.01				5315.69
		ADE	8204.67	1449	12.32	6	.06				5306.67
		AE	8213.69	1450	21.34	7	>.01	9.02	1	>.01	5313.69
		E	8313.08	1451	120.73	8	>.01	108.41	2	>.01	5411.08
	2	Saturated model	6697.19	1443							3811.19
		Constrained means	6702.76	1445	5.57	2	.06				3812.76
		Constrained variance	6702.96	1445	5.77	2	.06				3812.96
		ACE	6724.35	1449	27.16	6	>.01				3826.35
		ADE	6717.94	1449	20.75	6	>.01				3819.94
		AE	6724.35	1450	27.16	7	>.01	6.41	1	.01	3824.35
		E	6769.99	1451	72.80	8	>.01	52.05	2	>.01	3867.99
	3	Saturated model	8728.25	1443							5842.25
		Constrained means	8732.59	1445	4.34	2	.11				5842.59
		Constrained variance	8729.90	1445	1.65	2	.44				5839.90
		ACE	8739.73	1449	11.48	6	.07				5841.73

	ADE	8734.32	1449	6.07	6	.42				5836.32
	AE	8739.73	1450	11.48	7	.12	5.41	1	.02	5839.73
	E	8900.75	1451	172.50	8	>.01	166.43	2	>.01	5998.75

Notes:

-2LL – minus twice the log likelihood; *df*- degrees of freedom; *AIC* – Akaike's information criterion

For drive for thinness and bulimia at Time 2, MZ and DZ variances could not be equated, suggesting that there could be sibling interaction.

Table S7 - Common pathway model results with confidence intervals: genetic and non-shared environmental influences on the latent factor, and latent factor and time-specific influences on each variable

		Drive for Thinness			Bulimia			Body Dissatisfaction		
Etiological influences on the latent factor	A_l	.79 (.74-.84)			.60 (.49-.70)			.82 (.77-.86)		
	E_l	.21 (.16-.26)			.40 (.30-.51)			.18 (.14-.23)		
Time		1	2	3	1	2	3	1	2	3
Latent factor influences on each variable	L	.75 (.73-.78)	.91 (.89-.92)	.79 (.76-.81)	.49 (.44-.53)	.74 (.69-.79)	.69 (.64-.74)	.78 (.76-.80)	.93 (.92-.95)	.82 (.79-.84)
Time-specific etiological influences on each variable	A_s	.16 (.12-.20)	.00 (.00-.01)	.11 (.06-.16)	.15 (.09-.21)	.03 (.00-.10)	.12 (.04-.20)	.16 (.12-.20)	.00 (.00-.02)	.13 (.08-.17)
	E_s	.28 (.24-.32)	.18 (.15-.21)	.28 (.23-.33)	.61 (.55-.68)	.42 (.34-.50)	.40 (.32-.49)	.23 (.20-.27)	.13 (.11-.16)	.21 (.17-.25)

Notes:

A – broad-sense (additive and dominant) genetic effects; E - non-shared environmental effects; L – Latent factor.

95% Confidence Intervals (CIs) are presented in brackets. CIs not inclusive of zeros indicate significant influences. Non-overlapping CIs mean significant difference between the values.

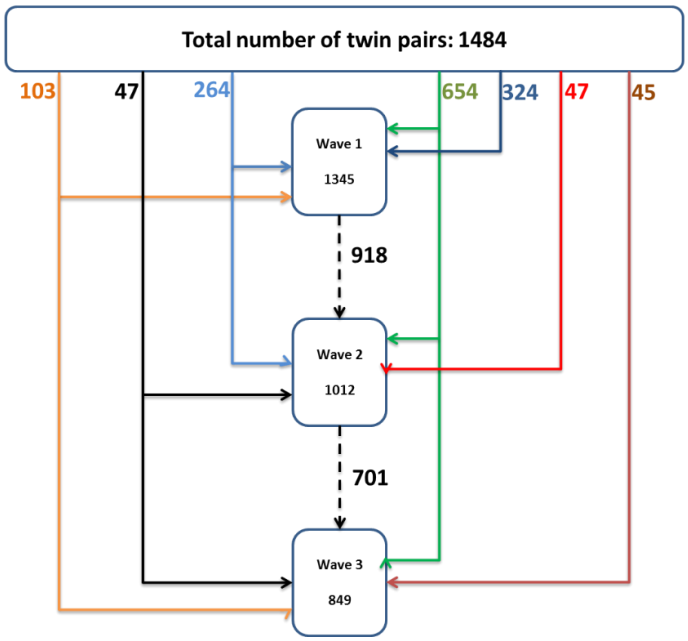
L needs to be squared to inform about the proportion of total variance accounted for by the latent factor. L^2 should be multiplied by A_l to obtain the proportion of the total variance due to the genetic influences from the latent factor. L^2 should be multiplied by E_l to obtain the proportion of the total variance due to the non-shared environmental influences from the latent factor. Total variance of a trait = $L^2 + A_s + E_s$

Table S8– Univariate AE results – non-overlapping age ranges

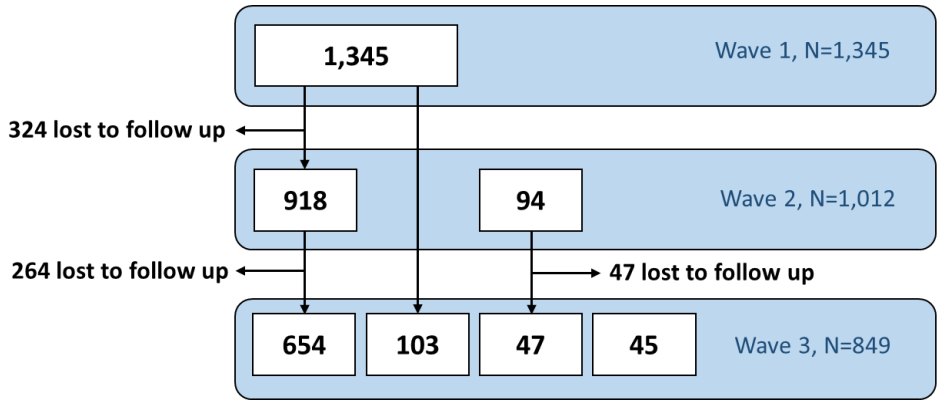
		A	E
Time 1 (14-15 years)	Drive for thinness	.54 (.45-.61)	.46 (.39-.55)
	Bulimia	.19 (.09-.28)	.81 (.72-.91)
	Body dissatisfaction	.64 (.57-.70)	.36 (.30-.43)
Time 2 (16-17 years)	Drive for thinness	.60 (.53-.66)	.40 (.34-.47)
	Bulimia	.39 (.30-.47)	.61 (.53-.70)
	Body dissatisfaction	.67 (.61-.72)	.33 (.28-.39)
Time 3 (18-19 years)	Drive for thinness	.63 (.54-.71)	.37 (.29-.46)
	Bulimia	.37 (.27-.47)	.63 (.53-.73)
	Body dissatisfaction	.71 (.63-.77)	.29 (.23-.37)

Figure S1 – Attrition and Participant flow

(a) Attrition



(b) Participant Flow



Notes:

1,345 families completed wave 1. Out of this group, 918 families took part at time 2, while 427 did not. Thus, the retention rate from time 1 to time 2 was 68.3%. Out of 427 twin pair that did not continue to

time 2, 324 were lost from the rest of the study, and 103 returned to take part at time 3. Furthermore, at time 2, 94 new families joined the study, making the total of 1,012 families in wave 2. Next, 701 families continued from time 2 to time 3, and 311 were lost to follow up, resulting in the retention rate of 69.3% from time 2 to time 3. Additionally, at time 3, 103 families from wave 1 took part, as well as 45 new families joined, making it a total 849 families. In total, 1,484 families took part in at least one time point, and 654 families took part in all three time points.

Attrition was not associated with baseline eating disorder symptoms.